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1. Perkins, J. L.: Kansas State M. J. (to be published).

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1. Phelps, W. M.: Arch. Pediat. 76:243 (June) 1959. 2. Spears, C. E., and Phelps, W. M.: Arch. Pediat. 76:287 (July) 1959.

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CLEFT LIP: EARLY VERSUS DEFERRED COSMETIC REPAIR

JOSEPH LUBART, M.D., F.A.C.S.*

New York

The incidence of clefts is commonly reckoned at about 1/1000[1] births but varies in different countries from 1/1,742 in England to 1/638 in Germany. In as much as the repair of a harelip involves the most prominent features of the face, the adult cosmetic result becomes a matter of great importance. It is the present teaching that the harelip should be repaired as soon as the patient's condition permits, certainly before the bones have lost their pliability. Some intervene within 48 hours of birth. Others prefer to wait until the child has regained its birth weight, while still others delay surgery until the infant weights 10 lb. i e. at two or three months of age. Swenson[2] in his pediatric surgery writes: "Early repair is now universally advocated, with some difference as to the actual age selected for operation. Repair 10-14 days after birth when the feeding schedule has been established and a search for obscure anomalies has been completed is preferred."

It is generally agreed, however, that the ultimate results of early cosmetic repair of the harelip deformity leave much to be desired. Fomon[3] *et al* have stated: "On the basis of our experience we are convinced that the degree of the deformity in the adult harelip nose is due more to the primary surgery than the embryonic fault (figs. 1 and 2)." This is in accord with my findings and also that the more satisfactory the cosmetic result in the infant, the more pronounced the deformity is likely to be at puberty. Although consideration of surgical technic is beyond the scope of this paper, it would be enlightening to know what the cosmetic correction involves. There is wide stripping of the soft parts, extensive V excisions or the use

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Fig. 1.—Harelip nose unoperated on, each part of face enlarging in "just the proper direction and amount to maintain the stabilization of the whole." Such a case offers no problem. (Courtesy Dr. Rambo.)

of complicated geometric flaps all of which is likely to damage the growth potential. In their recent research on facial clefts, Slaughter and Brodie[4] found that "Surgery can and does inhibit normal growth. This interference is directly proportional to the amount of injury to growth centers and to diminution of blood supply to the parts concerned." These workers also believe that growth in congenital deformities is similar to that in normal tissues, each part of the face enlarging in "just the proper direction and amount to maintain the stabilization of the whole." With the introduction of the Broadbent-Bolton[5] Cephalometer in 1931 it became possible to measure accurately the growth patterns of the head by super-imposition of a series of consecutive cephalometric roentgenograph tracings. By use of this method in cases operated on and unoperated on, Graber[6,7] concluded: "The evidence afforded by these comparisons would seem convincing proof of the damaging effects which can follow operations in early life, if tissues which should be free to expand and develop are confined and restricted in a vice of heavy unyielding suture material and subsequent scar-tissue formation."

Brodie,[8] Slaughter and Brodie,[4] Graber[6] and Broadbent[9] performed growth surveys which demonstrated that four-fifths of the

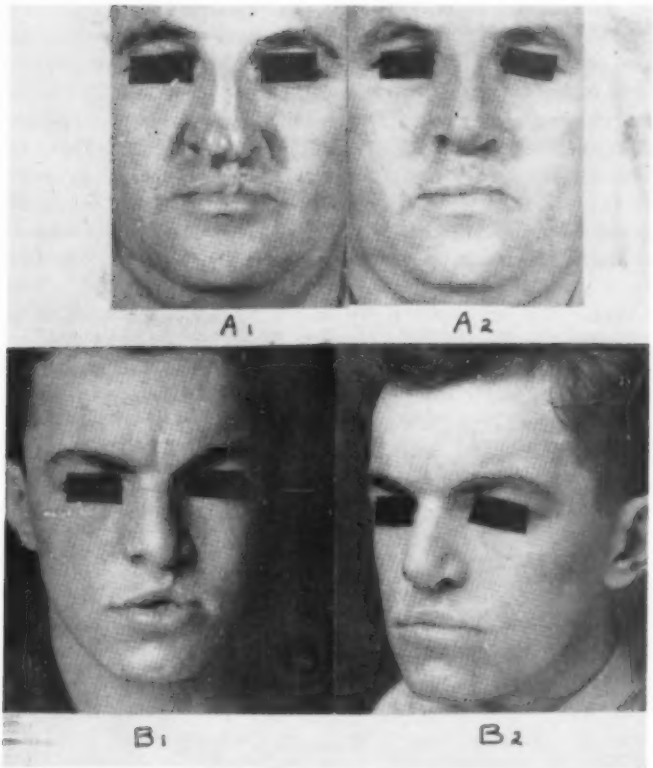

Fig. 2.—Repair of harelip nose: *A1*, result of physiologic adjustment and approximation in infancy, with no attempt at cosmetic result. *A2*, result of revision. Note absence of stigma of harelip deformity. *B1*, result of closure in infancy, with cosmetic objective. *B2*, result of revision. Note stigma of harelip deformity, scaphoid facies, and nasal disfigurement.

total facial growth takes place during the first five years of life, at which time the facial pattern is definitely established. More recently, using similar cephalometric methods, Ortiz-Monasterio[10] *et al* examined a group of untreated facial clefts and found that the forward growth of the upper maxillae is the same or greater than in normal cases. This is directly contrary to the frequently held theory that the underdevelopment of the face is due to the same embryonic factor that produced the cleft.[11] This would indicate that the "dish-face" deformity so frequently seen in these cases is caused by early

or repeated and aggressive surgery. It furthermore seems reasonable to suppose that if early surgery produces severe growth defects that surgery performed between 18 and 24 months will likewise produce them, though to a lesser extent.

The adult appearance, following such early surgical interference, is distinctive; the maxillary, nasal, ethmoid and malar bones are underdeveloped and displaced backward with resulting nasal asymmetries. Because of either the scar-tissue pull, damage to the growth potential, or pressure of the tight lip following the primary cosmetic operation, the upper jaw has come to lie inside the lower. The teeth are stunted, displaced and carious. The relative prominence of the forehead, the recession of the middle of the face, and the prognathic mandible give the person the characteristic "dish-faced" appearance of the harelip patient. Such deformities are obviously more easily prevented than corrected. It is well known among those entrusted with the secondary repair of the adult harelip nose that such surgery is often a frustrating experience.

In the infant the tissues are small and fragile and at this stage of life, a sacrifice of material no matter how slight represents a considerable loss later in life. Similarly an error in technic will be magnified many times over with the development of the face. Kazanjian[12] is authority for the statement that: "The excision of a piece of skin as small as one quarter of an inch in length is equal to half an inch in length in the adult." According to Blair,[13] "A sixty-fourth of an inch one way or the other will make a noticeable difference."

Despite this and other considerable evidence against the advisability of extensive surgery in infants, the consensus of surgical opinion is that expressed by Le Mesurier[14]: "The operation is done almost entirely for the sake of appearance and should be done with the object of making both the lip and the nose as nearly normal in appearance as possible at one operation." Although much has been written about this subject, no new principle in theory has been developed in this field in over a century. Whatever recent advances that have been made were merely in the refinement of technique and instruments; all for the purpose of obtaining some additional cosmetic benefit. What is obviously needed is a revised concept emanating from a reorientation of thinking. Theoretically, the policy of perfection would indicate the advisability of deferring the cosmetic repair of the harelip until the tissues are fully matured and the patient's cooperation is obtainable, to permit of careful, leisurely repair. It must therefore be a very powerful motivating force that

could persuade a surgeon, who would unhesitatingly delay a rhinoplasty until the parts have fully developed, to allow himself to do the extensive surgery necessary for a cosmetic harelip operation in the infant. Such a compelling force, however, must indeed be dealt with in the urgent demands of the parents; for a facial deformity in the newborn is, for them a devastating experience. Add to this the stigma of heredity and the calamity assumes catastrophic proportions. Aside from the effect on the parents one must also consider the complexes that are sure to arise from the ridicule and mimicry of the child's companions.

Admittedly these are delicate situations and require tactful handling on the part of the pediatrician. Consultation with a psychiatrist to weigh the psychologic damage inflicted by postponement of operation against the possible injury to growth centers, incurred in extensive early intervention, would be of great benefit in determining the optimal time for the lip reconstruction. The cooperation of the parents can usually be obtained by a frank and tactful explanation that although an immediate cosmetic closure is possible the longer the surgery is delayed, the better will be the ultimate result. The discussion with the parents must include the balancing of the short span of years from infancy to age 5 or 6 years, with the first part completely in the security of the home, against the remainder of the patient's biblical three score and ten.

There are other claims made for early closure. Among these is one that, "The operation facilitates feeding."[15] With modern harelip feeders adequate nutrition can always be maintained. Another argument is that, "In infancy the risk of life is under ordinary conditions very trivial indeed." It would be very interesting, in this connection, to have some definite information as to the shock-resistance and healing qualities of infants as compared with those in older children. We have been unable to find such statistics. A more valid reason is the claim that early closure exerts a beneficial effect on the future development of the parts. It is assumed that in a normal infant the counterpressure of the lip, tongue and cheek molds the alveolar process. Since in a cleft lip the labial forces are lacking, the tongue tends to push forward the alveolar arch, while the buccal forces constrict the lateral segments, resulting in a cross bite. The claim is made that not only does the operation restore the labial forces but in complete clefts the elastic tension of the repaired lip favors approximation of the alveolus. When done late, repositioning

of the premaxilla proves more difficult owing to the decreased pliability of the bone. Granting this is so, the beneficial effect of the elastic pressure of the reconstructed lip can be obtained by limited initial surgery designed merely to restore this function without the extensive trauma, sacrifice of tissue and growth impairment necessary to obtain a cosmetic result at this stage of life. Another even more conservative method is by use of a prosthesis such as the one designed by McNeil,[16] Senior Lecturer at the University of Glasgow, for retropositioning of the premaxilla and the expansion of the dental arch (fig. 3).

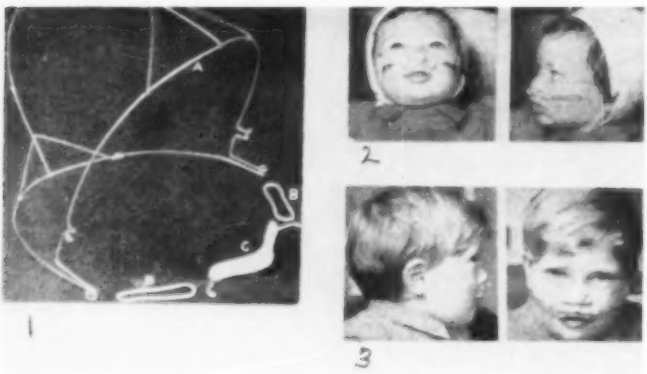

Fig. 3.—McNeil's prosthesis: (1) Components of appliance: *A*, stainless-steel harness; *B*, rubber bands; *C*, labial attachment. (2) Frontal and lateral views of patient at 10 months. (3) Frontal and lateral views of patient at 2½ years. (Reproduced from McNeil: Oral and Facial Deformity, Sir Isaac Pitman & Sons, Ltd.)

SUMMARY

The purpose of this paper is to enlist the aid of the pediatrician in a more realistic determination of the optimal time for the correction of the harelip deformity. The pediatrician is in the best position to evaluate this problem since he alone follows the patient from birth to puberty.

We are convinced that attempts at a cosmetic closure of the harelip in infancy with its attendant wide stripping of the soft parts, exensive V excision, or the use of complicated geometric flaps and consequent damage to growth potential, are in large measure the cause, in later life, of the nasal asymmetries, recession of the maxilla, malocclusion and retrussion of the upper lip, giving the characteristic "dish-faced" appearance. Our experience has shown that the

degree of the deformity in the adult harelip nose is due more to the primary surgery than to the embryonic fault. A facial deformity in the newborn is a devastating experience to parents but their importunities for early cosmetic repair must be resisted by the pediatrician and countered with a frank and tactful explanation that such surgery at so early an age is to be had only at the expense of future facial development. They must be told that the longer the cosmetic surgery is delayed, the better the ultimate results. An initial simple closure is all that should be advised at most with the cosmetic repair postponed until the facial pattern is fully developed as in all other reconstructive surgery. A suitable compromise between the psychologic hazard of late repair and the hazard to facial development of early cosmetic correction might consist of simple closure of the cleft without sacrifice of tissue at about one year of age with definitive surgery being performed at 5 or 6 years of age to avoid the psychologic trauma that might otherwise occur during attendance at school.

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OTOLOGICAL FINDINGS IN BIRTH INJURIES OF THE NEW BORN*

Cranial and Cerebral Lesions in the Newborn and Their Pathogenesis—Part I

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I. Experimental Investigations

For over a hundred years it has been well known that the presenting part of the fetus may suffer injury as a result of the birth process itself, but for a long time the prevention of such injury and its sequelae received little attention. At first it was assumed that these injuries were rare and that the causes were of purely mechanical nature, such as narrow pelvis, long and difficult labor, use of forceps, and the like. Not until Seitz in 1907,[80] Benecke in 1910,[9] and, somewhat later, Ylppö[106] reported the results of their investigations did clinicians become interested in the problem of birth injury. All three had stressed that difficult labor or the use of forceps were not the only factors responsible for intracranial injury during birth.

Warwick[102] found intracranial hemorrhages in 18 of a series of 36 cases of stillbirth and neonatal death, and some workers, Holland[44a] among them, even asserted that cerebral birth injury was one of the most important causes of neonatal death. However, attention was directed mainly to the symptomatology of birth injury rather than to the mechanisms of the injury or its later sequelae. The whole problem was brought into proper focus when Schwartz, in 1921, reported his postmortem findings in a large series of neonatal deaths. He formulated a theory on the true causes of the damage suffered during birth, and described in detail the acute and chronic intracranial changes and the resultant anatomic effects. Since then, he has repeatedly discussed the wide-ranging problems, has provided additional data to support his theory, and has succeeded in convincing early opponents of his views. He clearly disapproved that intracranial hemorrhage in the newborn is due to congenital syphilis, or to congenital hemorrhagic disease resulting from vitamin K deficiency, to anoxia, asphyxia, or toxemia.

* This paper is based on our own experimental and clinical investigations and is supplemented by the more recent literature.

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MECHANISMS OF BIRTH INJURY

The head is the presenting part in 97 per cent of all births, and is therefore most likely to be injured during birth. If arm, buttocks, or back is the presenting part, that too may show injury. According to Schwartz,[77] there is some degree of injury of the presenting part in 65 per cent of all newborn infants.

Schwartz believes that hemorrhage in the newborn is caused by the difference between the lower atmospheric pressure on the presenting part and the higher pressure on the intrauterine part of the fetus. So long as the fetus is within the intact amniotic sac, it is under uniform pressure, which during birth ranges from 80 to 250 mm. Hg. As soon as the sac bursts, and the emerging parts enters the external uterine orifice, it closes the orifice like a ball valve, and is exposed to atmospheric pressure while all the rest of the fetus is still under the higher intrauterine pressure. The effect can be illustrated by applying a suction cup to the skin. The rapidity of the swelling which follows is governed by the degree and length of time during which the site is exposed to the negative pressure within the cup. So long as the suction is maintained, fluid accumulates in the tissues and blood vessels of the area covered by the cup, the rim acting as a sort of tourniquet and the negative pressure preventing dispersal of the accumulated fluid.

In much the same way, the rim of the external uterine orifice acts like a tourniquet around the presenting part, which may be compared to the area covered by a suction cup. Body fluids are pulled into the exposed part and cannot flow back. Furthermore, the pressure difference operates so long as the presenting part closes the external uterine orifice like a ball valve. Clinically therefore, this means until the infant is born.

Experimental data have confirmed Schwartz's assumption that such a pressure differential, if maintained for any length of time, must result in vascular distention, stasis, and, finally, in vascular tears in the area under the lower pressure. The mechanism he postulated would clearly explain the occurrence of caput succedaneum and cephalhematoma. Any significant difference between the pressure in the uterine cavity and the atmospheric pressure on the head must cause vascular stasis and rupture of scalp veins. Since the dural veins, venous sinuses, and pial and cerebral veins are directly connected with the scalp veins, longitudinal sinuses, and large and small fontanels, stasis and rupture of the deeper lying intracranial structures can occur easily.

Schwartz injected colored fluids into the cranial blood vessels of cadavers and found that the fluids appeared in those cerebral sites where anatomic changes had occurred. His experimental findings and the fact that the typical cerebral hemorrhage in the newborn occurs in the vascular system of the great vein of Galen, led him to conclude that such mechanical causes of stasis and vasomotor disturbances explain most cases of cerebral injury in the newborn. In his opinion, deformation of the skull, shearing effects, or other stresses to which the skull may be subjected during birth play a minor role in cerebral birth injury.

Experimental Evidence

Newborn rabbits, dogs, and cats apparently do not have birth injuries, since no cerebral hemorrhages, necrotic foci, or areas of softening are found in these species.[10,85,86,90] In our investigations we confirmed this, but the petrous bones of the animals revealed small hemorrhages closely resembling those reported by Voss,[98,100] to be present in newborn infants. The occurrence of hemorrhages in the petrous bones and eyes was related to the manner in which the animals were killed. Middle and inner ear hemorrhages were most severe in chloroformed animals, moderate in those killed by cardiac section, and slight in those killed by cutting the throat. Slight hemorrhages were also found at characteristic sites in the head and in many other organs in fetuses of cow, sheep, and pig at various stages of development. However, these could only have been a terminal event, since anoxia quickly causes fetal death after maternal sacrifice. In our study of Hortega's cells in various species in relation to cerebral injury, we also found that the minimal hemorrhages in the ears were a terminal event. It is noteworthy that these terminal hemorrhages in the fetus are slight, whereas in the newborn animals the experimentally induced hemorrhages were massive.

Technic. A suction cup with manometer was applied to the heads of 7 puppies, 9 rabbits, and 5 kittens; most of the 21 animals were newborn, while a few were 24 to 48 hours old. A vacuum was created within the cup so that there was suction on the area covered by it. The negative pressure ranged between one-twentieth and one-third atmosphere. In the first few animals, the scalp was incised and the cup applied directly to the bone, since uniform placement of the cup over the small or large fontanel was difficult because of the loose scalp. Thereafter, the cup was applied to the intact scalp, with the greatest suction occurring over the longitudinal sinus.

The negative pressure was increased slowly, kept at a high level and then dropped to normal, or the whole procedure was several times repeated for 2, 3, or 5 hours. In some animals, after the negative pressure reached a high level, it was maintained for several hours. In our opinion, the latter more closely resembles the physiologic conditions in human birth. Many of the animals died within a day or two; the others, with the exception of 2 kittens, who were sacrificed after 3 weeks, were killed 2 or 3 days after the end of the experiment. The petrous bones of all animals were fixed in the usual way, embedded in gelatin, and examined by frozen section.

Results. The changes regularly observed resembled caput succedanum, cephalhematoma, and the skull deformations found in newborn infants. There were extensive thromboses in the region of the longitudinal sinus in 4 animals, in the cavernous sinus in 1, and in the rectus sinus in 1. In several animals, pial, intraventricular, and intracerebral hemorrhages were present, identical in site and character with those in newborn infants with birth injury. Small foci of cerebral necrosis were noted in 2 animals 3 days after injury.

A study by Ricker and Regendanz (1921) of the effects of vascular lesions in general is of interest in connection with our experimental findings. They found that tissue injury or irritation decreased the blood flow, in some cases until it ceased completely. The circulatory disturbances led to infiltration of the tissues by plasma, erythrocytes, and leukocytes, to proliferation of the reticular cells, and to parenchymal degeneration or complete destruction if the disturbances lasted long enough.

Our results in the first 12 animals were reported in 1924[10]. We had found typical truamatic lesions, such as Schwartz had observed in newborn infants: cranial, periosteal, pial, and cerebral hemmorhages; thromboses of the longitudinal and rectus sinuses and their intracerebral tributaries; in 2 animals, fresh necrotic foci in the white matter of the frontoparietal and occipital lobes.

The petrous bones showed extensive and marked vascular disturbances, like those which Voss[97,98] found in newborn infants: (*1*) engorged arteries and veins; (*2*) extensive hemorrhages in the middle ear cavity; (*3*) congestion, hyperemia, and hemorrhages in the middle ear membranes, bone marrow, semicircular canals, cochlea, modiolus, spiral ganglion, basal membrane, and facial and acoustic nerves. The hemorrhages in the semicircular canals almost invariably originated from the blood vessels of the membranous canals and spread into the perilymphatic space in raylike fashion; only rarely was blood present in the endolymphatic space.

The hemorrhages were equally severe in both petrous bones in almost all the animals; in one animal there was also a severe hem-

orrhage in the nasal cavity and nasal membranes as well. An acute otitis media was found in 2 animals, in addition to the hemorrhage. Otitis media occurs commonly in laboratory animals[17]; it is therefore unlikely that the hemorrhages in the tympanic cavity caused the inflammation. At most, they may have provided a favorable condition for its occurrence.

The same pathologic features were found in the petrous bones of the 2 kittens sacrificed after 3 weeks. A control kitten from the same litter, killed at the same time, showed no pathologic features. The erythrocytes in the perilymphatic and endolymphatic spaces were intact, probably because the fluid in these spaces acts as an isotonic solution and preserves the red cells. It is noteworthy that similar findings in the labyrinth are common after skull fracture or concussion. In another experiment, negative pressure on the ear lobe of the guinea pig was created by suction. Hemorrhages were found in the inner ear on that side, and may well have been the direct result of tears in the blood vessels by the negative pressure. But the aural hemorrhages resulting from the negative pressure on the head could only be secondary to disturbances in the intracranial venous circulation. Suction (i.e., negative pressure) on the head results in congestion and stasis in the intracranial veins, the superior longitudinal sinus, and the small and large fontanels, then proceeds to the transverse and sigmoid sinuses, advances to the superior and inferior petrosal sinuses, and from there spreads into the petrous bone and the tributary veins. If the negative pressure is high and of long duration, the congestion and stasis in the small veins becomes severe enough to cause hemorrhages by diapedesis or through vascular tears.

Ylppö (109) investigated the mechanisms of hemorrhages in the newborn. He applied a suction cup to the body and extremities, and found that the resultant hemorrhages occurred more easily, more often, and more extensively in the premature than in full-term infants. The reason he suggested, was the greater fragility of the vascular walls in premature infants. Schwartz had also noted the greater frequency of cerebral birth injury in premature infants.

Metzger (59), using our methods, produced retinal hemorrhages in newborn animals; he ascribed them to circulatory disturbances in the region of the longitudinal sinus. Thielemann (92) confirmed the occurrence of lesions in the inner ear due to birth trauma. Guttner (42), using guinea pigs, tried to simulate the birth process as closely as possible. A large glass jar with a narrow neck represented the uterus, a rubber bag within it the amniotic sac, and the animal within the bag the human fetus.

As the pressure inside the jar (i.e., the intrauterine pressure) was raised, the animal's blood pressure and cardiac function were kept under observation. So long as the rubber bag was intact, the increased intrauterine pressure affected neither blood pressure nor cardiac function; as soon as the bag was torn and the head emerged into the neck of the jar (the pelvis), both blood pressure and cardiac function were affected. Unfortunately, Guttner did not mention whether he had found any cerebral lesions.

STUDY OF INTRACRANIAL VASCULAR SYSTEM

The results of our animal experiments led us to study the venous connections between the petrous bone and the large venous sinuses and cranial veins in the cadavers of several children and a few adults. The existence of a direct connection between the scalp veins and the longitudinal sinus, the smaller sinuses, and the dural veins, as well as between the ocular veins and the large intracranial veins and sinuses, is well known.

Some of the early investigators had even realized, on the basis of the spread of infections from the middle to the inner ear and contiguous bones, or vice versa, that complete drainage of the petrous bone is accomplished by small veins in the petrous bone emptying into the superior, inferior petrosal, or sigmoid sinuses or into the jugular vein or bulb. These venous connections play an important part in the spread of otogenous infection with thrombosis of the lateral or cavernous sinus or in the development of meningitis and brain abscess.

In our circulatory study we used a mixture of a 12 per cent gelatin solution and 12 to 15 parts by weight of red oxide of lead. The course of the mixture can be followed by the naked eye; it is in a fluid state at 97 F., solidifies a few minutes after injection, and does not dissolve thereafter. The brain or petrous bones can therefore be removed without spillage of the material from the blood vessels. After removal, brain or bone was fixed in formaldehyde and later embedded in gelatin in the usual manner; frozen sections were then made. Some were stained with hematoxylin, others were left unstained; all were examined in transmitted or incident light. Various other substances were tried but were less satisfactory. Radiopaque materials gave such a confused roentgenographic picture that clear interpretation was impossible.

Because in the adult cadaver the top of the skull had to be removed in order to reach the large fontanels or sinuses, so that the injected material escaped before it could reach the small veins in the petrous bones and in the pituitary gland, our study was much more successful in the young. In the latter, the material was injected directly into an intact vascular system via a fontanel without removing the skull cap.

The injection was completely successful in 12 young and 3 adult cadavers. After a single injection into the longitudinal sinus, the material was seen to spread directly by way of the transverse and

sigmoid sinuses into the jugular vein and to fill the pial and cerebral veins draining the longitudinal sinus. The material also filled the superior and inferior petrous sinuses, the veins coming from the petrous bones, the cavernous sinuses, and the numerous orbital blood vessels. We thus demonstrated a communicating vascular system, with the longitudinal sinus as its main source.

On microscopic examination, the injected material was found in the branches of the internal auditory vein, the vein of the vestibular aqueduct the cochlear canaliculi, the veins in the membranous semicircular canals and in the middle ear membranes, and in the bone marrow. When injected with stronger pressure, the material escapes into the endolymphatic and perilymphatic spaces through tears in the finer veins and venules. We clearly demonstrated the existence of small veins leading directly from the petrous bones to the superior petrous sinus. The same venous distribution was found in the adult cadavers.

Of particular interest were the findings on the vascular supply of the pituitary gland. As is well known, the veins of the anterior lobe drain into the circular sinus between the anterior and posterior lobes or into the anterior and posterior intercavernous sinuses, those of the posterior lobe drain into the posterior cavernous sinus. The anterior lobe has a better arterial supply than the posterior lobe.

When the material was injected into the jugular vein (retrograde) or the longitudinal sinus, it spread not only into the dural veins, all the sinuses, and the cerebral and ocular blood vessels, but also into all the branches of the cavernous sinus and the vascular system of the pituitary gland. On histologic examination, the material was found in all the pituitary capillaries, particularly of the anterior lobe. The drainage of the gland therefore is part of the closed intracranial venous system. This fact is particularly significant, since according to Schwartz the longitudinal sinus, as a part of the closed intracranial venous system, is most subject to negative pressure during birth.

VASCULAR SYSTEM OF PITUITARY GLAND

From the few reports on the pituitary glands in the newborn,[56] it was known that iron pigment had often been demonstrated in the posterior lobe, that hyperemia and hemorrhages had been found, and occasionally necrotic foci, sometimes with calcification. The hyperemia and hemorrhages were ascribed to skull injury or brain tumor. There are also reports of hyperemia and hemorrhage in the

pituitaries of anencephalic infants and in cretins. Simmonds[87] found necrotic foci and gummas in the pituitaries of 12 newborn infants with congenital syphilis, and ascribed them to the syphilitic infection. Others confirmed his findings, as for example Schmitt,[74] who found necrotic foci in 3 of a series of 34 cases of congenital syphilis. However, these foci were ascribed to syphilis only in the newborn; the necrosis found in adults was always held to be due to thrombophlebitis or arteriosclerosis. Was then the necrosis in syphilitic children the result of syphilis, or of another reason, for example, birth injury? To obtain an answer I did the following:

Histologic examination of serial sections of the pituitary glands of 40 nonsyphilitic newborn infants revealed a vascular hyperemia in the anterior lobe in 32 of them: (*1*) The capillaries were filled to bursting, especially those between the anterior and middle lobes directly beneath the dura. (*2*) The distended capillaries radiated from the center toward the periphery, where the distention was greatest. In some pituitaries, the hyperemia was so severe that the cellular trabeculae between the capillaries had more or less atrophied, so that the capillaries lay next to each other or were separated by foci of necrosis (3 cases). Around such foci there was severe hyperemia, and frequently extravasation of blood. A much less severe hyperemia was found in the posterior lobe (3 cases only), but in 1 of them an extensive necrosis in the center of the lobe was present. On the basis of our findings, therefore, conclusions by earlier investigators that congenital syphilis was responsible for the necrosis seems unfounded.

Since we did not examine the pituitaries of normal older children and adults, or those with such conditions as myxedema, dystrophia adiposogenitalis, or diabetes mellitus, we draw on the reports of other investigators. Eichhorst[30] reported necrotic foci, hyperemia, iron pigment, atrophy, and connective tissue proliferation in the anterior lobe of an older child with myxedema and dwarfism. Similar findings have been reported by a number of others, who believed the pituitary lesions to be primary cause of such conditions as eunuchoid dwarfism, myxedema, idiocy with obesity, infantile diabetes, dystrophia adiposogenitalis and obesity. The pituitary also plays a primary role in the classic picture of Simmonds' disease.[16,87,88,89]

On the basis of what is known about the vascular system of the pituitary gland and the intracranial venous system, it may be concluded that certain changes in the pituitary gland of the newborn—hyperemia, parenchymal atrophy, and necrosis—can result from

birth injury. Furthermore, some disorders in older children may be late sequelae of pituitary injury during birth.

LIPIDS IN CENTRAL NERVOUS SYSTEM

The problem whether the lipids found in the brains of newborn animals are physiologic or pathologic has long been the subject of a lively discussion. We therefore tried to establish, if possible, whether (*1*) lipid is deposited at any time during the development of the central nervous system of various species, (*2*) the lipid cells in the central nervous system of newborn infants is identical with that of animals, (*3*) the lipid tissue elements are related to the development of the myelin sheath, (*4*) lipid is present in Hortega's cells during their development, (*5*) the location of Hortega's cells is related to the lipid-containing cells, and (*6*) the morphology of Hortega's cells resembles that of the lipid-containing cells. A detailed study was made of the brains of 17 fetuses of sheep and 7 of cows at various stages of development, of 6 newborn rabbits, of 6 rabbits 2 to 6 days old and 2 rabbits 4 weeks old, and of 6 puppies 8 days old and 3 puppies 5 weeks old. In addition, the brains of 3 puppies 8 days old and of 2 rabbits 4 weeks old were injured by insertion of a sharp needle into the brain substance and the animals were sacrificed a few days later. As a final control, we used the brains of 5 newborn chicks.

The fat-staining methods then available (Aschoff-Kawamura) showed no lipid-containing cells in the central nervous system of the 47 animals with uninjured brains. This confirmed earlier concepts[77,84,90,95] that the central nervous system of man develops without the deposit of lipids in the system itself. Fatty degeneration was found in the 5 injured brains which differed in no way from that found by Schwartz[77] in newborn children with birth injury. The lesion consisted of isolated, hypertrophic, glistening, lipid-containing glia cells, some of them containing lipoid inclusions and staining with Hortega's stain.

Lipid-containing cells may therefore be assumed to be of pathologic significance and an indication of central nervous system damage. The cells which Hortega described to be present in cerebral tissue do not contain lipid and do not resemble the granular, fatty cells in the brains of newborn children noted by Virchow[94].

SUMMARY

(*1*) Hemorrhages were produced in the middle and inner ears

and the brains of experimental animals (dogs, cats, rabbits) by means of negative pressure applied in the region of the longitudinal sinus. Occasionally, thromboses in the venous sinuses and necrotic foci in the brain were present.

(*2*) The hemorrhages were the result of the congestion caused by the negative pressure at the superior longitudinal sinus. Their spread through the intracranial venous system led to the hemorrhages, thromboses, and necrotic foci in the brain, pituitary gland, ears, and eyes. The severity and extent of the congestion and stasis and the resultant disturbances in the intracranial venous circulation depended on the duration and degree of negative pressure.

(*3*) Intracranial injection of a mixture of gelatin and red oxide of lead into cadavers of children and adults proved that the intracranial venous system is a completely communicating closed system. It also proved that the venous drainage of the pituitary gland via the cavernous sinus is closely related to the large dural sinuses, thereby exposing the gland to birth injury.

(*4*) Lipid-containing cells are not present in normal animal fetuses, but do appear in young animals after experimental brain injury. These cells closely resemble those found in the brains of infants with birth injury.

(*5*) Our experimental and anatomic findings confirmed the fundamental observations of Schwartz,[77] who first described the cerebral lesions due to birth injury, and the observations of Voss[97], the first to report aural lesions in the newborn infant.

II. Clinical Investigations

The experimental data and the pathologic-anatomic findings in the newborn inevitably led to the question: Could the clinical features produced by the type of birth injury discussed be recognized at birth, and might not this injury and its symptomatology be masked by such common diagnoses as atrophy, asphyxia, intoxication, debility, and hereditary and constitutional noxae? Obviously, intrauterine damage, or damage of the genetic anlage, maternal infectious diseases, constitution, and heredity are responsible for some of the disorders. But are all cases of debility, deafness, otitis media, dementia, idiocy, paraplegia, Little's disease, epilepsy, convulsions, and facial and abducens nerve palsies to be ascribed to these causes? It is true that birth injury of the parathyroid and thyroid glands, the adrenals, testes, lungs, pancreas, kidneys, spleen, and parotid gland have all been reported. This cooperative clinical study was

undertaken by the Pediatric, Obstetric, Eye and Ear, and Pathology Departments of the University Hospitals in Frankfurt, Germany. Here we shall discuss only the head injuries during birth. No comprehensive clinical picture of such injury existed, and for analogies we had to draw on what was known of skull fracture and concussion. Under the leadership of Otto Voss, all of the investigative technics then in use for cerebral concussion were applied to the clinical investigation of intracranial birth injury.

Technic

A total of 190 persons (93 newborn infants, 97 ranging in age from 6 days to 21 years) were examined for general condition; blood pressure; pulse rate; temperature; weight; feeding; central nervous system motor disturbances; pareses or paralyses of the facial, abducens and oculomotor nerves; paresis or paralysis of the extremities; reflexes (Achilles tendon, Gordon's, patellar, cremasteric, abdominal); signs (Babinski's, Chvostek's, Oppenheim's, Trousseau's); and the state of the ocular fundi. The results of the ear, nose, and throat examination and the presence of spontaneous nystagmus were considered to be of particular importance. Almost all infants were given a hearing test with tuning forks and Bárány's noise machine, to establish if possible the presence of hearing function and of the auropalpebral reflex. Labyrinthine function was examined by irrigation with ice-cold water, with warm water (120° F.), and by Bárány's rotatory test. The latent time of the nystagmus, duration and intensity of the eye and head movements during and after rotation, eyeball deviation (an equivalent of nystagmus) eyelid and head nystagmus, and frowning and other facial movements were all noted.

In addition, the postural (otolith) reflexes, such as the tonic labyrinthine and labyrinthine righting reflexes, and the compensatory eye positions were tested. These reflexes had been found in decerebrate monkeys by Magnus and de Kleyn[57,58]. The tests were therefore thought to be indicated in the newborn because birth injury can produce cerebral conditions closely resembling decerebration in the experimental animal. Voss[97] reported the results of these special tests in detail.

We soon found that the usual neurologic tests were noninformative, so that the otologic and labyrinthine tests assumed special importance since they clearly indicated the presence or absence of cerebral trauma. A spinal tap was done in many cases, particularly in premature infants. The presence of blood in the cerebrospinal

fluid was an indication of hemorrhage. When there was reason to suspect more severe bleeding in the region of the terminal vein, pneumoencephalography was also performed.

Results

We separated our material into 4 groups: (*1*) 28 infants with a birth weight up to 1,800 Gm.; (*2*) 32 infants with a birth weight of 1,800 to 2,500 Gm.; (*3*) 33 infants weighing over 2,500 Gm.; and 97 children and adults with various defects, aged 6 days to 21 years. The first 2 groups were considered as premature.

Group 1. Of the 28 infants, 21 were first-born and 7 were second born. Labor ranged between 4 and 36 hours. In 20 of the births, the head was the presenting part; 1 was a breech presentation; 2 were delivered by forceps; and 2 by cesarean section. Most of the infants were examined on the day of birth; the remainder within 14 days of birth. All of the 10 infants who died 8 to 20 days after birth were examined post mortem.

The *general symptoms* were disturbances of the respiratory center—superficial respiration or signs of asphyxia. The drawn, mask-like appearance, the facial pallor, and the cyanotic skin were striking. In many cases the asphyxia was acute, and increased the cyanosis. Almost all of the infants had an abrupt, superficial respiration, sometimes fast, sometimes slow, interrupted by sudden crying. The sucking reflex was faint. The infants took food poorly, and catheter feeding was necessary in most cases. Spontaneous vomiting was rare. Singultus and yawning occurred frequently. The infants were in a stuporous state, but were easily frightened; a touch on the blanket caused a cry, with a quick return to stupor. A slowed pulse rate was found in a few. Weight decreased rapidly and sharply. The temperature was markedly subnormal. There was pronounced muscular rigidity, decreased skin turgor, and, in many infants, a scleroderma.

The most prominent *focal* symptom was convulsions. They occurred suddenly, sometimes when the infant was touched but often without any provocation. For the most part, they were brief, and in many recurred frequently; in a few, they recurred at lengthy intervals. In some the entire musculature was involved; in others, clonic jerks of the facial muscles or tonic-clonic contractions of the arm and leg muscles; concurrently, there was nuchal rigidity, the head was dug into the pillow, respiration was arrested, and cyanosis, foaming at the mouth, lockjaw, and oscillating movements of the

eyes occurred. The convulsions involved unilateral paresis. The entire body, or half of it, or one or more extremities, or part of the face, for example an area innervated by one or another nerve—facial, trigeminal, or glossopharyngeal. Some convulsions stopped suddenly, others slowly.

A unilateral facial paresis was another focal sign found in infants who had not been delivered by forceps. None of the infants had myosis or midriasis. Nuchal paresis and athetotic movements were present in many of the infants. Förster[35] ascribes such movements to injury of the corpus striatum, and Schwartz[76] found hemorrhages in the corpus striatum in such cases.

Spontaneous horizontal nystagmus lasting 4 to 8 weeks and for the most part of the constant type was present in 25 out of 28 (89.2%) infants; in most it was already evident during the first day of life, in a few within the first week. Usually the nystagmus was apparent when the infants were in horizontal position, but in a few cases movement of the eyeball to the opposite side could be elicited by turning of the head to one side. The nystagmus was bilateral toward the right or left in 50 per cent, but not equally pronounced to both sides in some. In 2 of the 3 infants without spontaneous nystagmus, caloric stimulation elicited no response and the rotary excitability was below normal.

Convergent or divergent strabismus of 6 to 8 weeks' duration was seen in many of the infants immediately after birth, and in a few appeared after some weeks.

A reaction to Bárány's noise machine was not evident during the first week of life, and often not until the third or fourth week. In contrast tests with high-pitched tuning forks elicited the auropalpebral reflex immediately after birth. Rarely, there was no acoustic reaction whatever in the first few days of life.

Irrigation with ice-cold water produced unquestionable eyeball deviation in 21 infants, bilateral nystagmus in 1 infant, and unilateral nystagmus in 1 infant. Immediately or a few seconds after the irrigation, the eyeballs turned toward the irrigated side and remained in the end position for 25 to 40 seconds. In some the eyeball on the irrigated side turned first, followed by the other eyeball. Spontaneous nystagmus continued in some infants, despite the eyeball deviation after irrigation. In most cases both the eyeballs returned to normal position simultaneously; in some there was a lag, the eyeball opposite the irrigated side returning to normal position first. Repeated caloric stimulation produced no reaction in 5 infants; in the 3 who died during the first week of life, autopsy disclosed severe cerebral injury.

Only a few infants responded to irrigation with water of 120° F. As in adults, this test seems not to be reliable enough.

The results after 10 turns in the rotating chair, with the infants in upright position, were: *1* Opposite turning of head and eyes as soon as rotation began. *2* Prompt turning of the head in the direction of rotation as soon as rotation stopped. *3* When nystagmus appeared, it did so immediately after rotation in the opposite direction to the rotation; it was always horizontal and lasted 12 to 25 seconds in about 50 per cent of the cases. Head turning was usually of shorter duration than eyeball deviation or nystagmus. *4* When eyeball deviation was the only reaction, it was in the direction of rotation, and lasted 20 to 25 seconds.

With the infant on its right or left side and the head forward, the head and eyes bent toward the feet during rotation; when rotation stopped, the head stretched backward and the eyes followed. The nystagmus was vertical (toward the feet), and the eyeball deviation in the direction of rotation (upward or headward). With the feet, instead of the head, in forward position, the reactions were correspondingly reversed. In this test, too, the head movement was of shorter duration than the eyeball deviation or the nystagmus; but in every case the two latter were of shorter duration than in the upright position (average, 6 to 10 seconds). Nystagmus in the side position was elicited in about half the cases.

Rotation of the infant recumbent on the back with the head forward resulted in turning of the head and eyes to the opposite side during rotation, with prompt turning in the direction of rotation as soon as it was stopped. The nystagmus, seen in about half the cases, was horizontal and opposite to the direction of rotation. Eyeball deviation was in the direction of rotation. Average duration of nystagmus was 10 to 20 seconds, or deviation 20 to 35 seconds.

Nystagmus of eyeball deviation was elicited in all the infants in this group in all 3 positions in the rotating chair. Many showed the experimentally induced nystagmus on the first day of life, while others only deviation of the eyeballs. The latter responded to the rotating chair test with deviation even in their third or fourth week.

In several infants, during or after rotation in one or another of the 3 positions, showed wrinkling of the forehead, head nystagmus, and eyelid nystagmus were also noted.

Slight postrotatory reactions were seen only in those infants who had no caloric reactions and died early.

Careful testing of the other reflexes contributed little information; even the infants with severe cerebral damage had few pathologic

reflexes. Voss's[90] detailed description of the tonic labyrinthine, the labyrinthine righting, and the neck reflexes make unnecessary further details here. We need only state that they were abnormal in many of the infants in this group.

In *summary*, in 28 infants of a birth weight up to 1800 Gm., the general symptoms dominated the clinical picture. Particularly striking was the frequency of a horizontal spontaneous nystagmus or eyeball deviation, the negative caloric response and rotatory hypoexcitability.

Group 2. The 32 premature infants (birth weight, 1,800 to 2,500 Gm.) were all examined on the day of birth or by the end of the first week. Most were first-born; labor lasted from 2 to 36 hours. Birth was spontaneous, with head presentation, in 30 cases; 1 infant was delivered by forceps, and 1 by cesarean section because of placenta praevia. Four died within 8 to 14 days, and one after 6 weeks. Follow-up for up to 6 months was possible in 10 infants.

In this group, too, the same general symptoms as in Group 1 were predominant. But the *focal* symptoms were more pronounced. Cortical convulsions and athetotic movements were more frequent and more severe. Spontaneous horizontal nystagmus, often with a rotatory component, was present in 24, and in 3 infants there was a vertical nystagmus. In other respects, the nystagmus was like that of Group 1 but disappeared after 6 to 12 weeks. Only 5 infants had no spontaneous nystagmus; 1 of them had been delivered by cesarean section, and in 2 the caloric and rotatory reactions were normal. The latter died of severe cerebral injury. Many of the infants had convergent and divergent strabismus. The results of all hearing tests were the same as in Group 1.

In the test with ice-cold water, the nystagmus or eyeball deviation lasted, on the average, 30 to 50 seconds. Eyeball deviation was the response of 66 per cent; of the 4 infants who did not react to the test, 3 soon died and autopsy revealed cerebral injury; the fourth, with extremely severe focal and general symptoms, responded to caloric stimulation for the first time at the age of 9 months.

The results of the rotation test in all 3 positions were the same as in Group 1. Postratatory nystagmus of 30 to 50 second duration occurred in 66 per cent. Eyeball deviation was the only response of 8 infants when first tested, but on reexamination some weeks later a typical nystagmus was elicited. Head and eyelid nystagmus, as well as wrinkling of the forehead was seen in many infants.

The neurologic examination in this group, too, was noninformative.

Group 3. Consisted of 33 infants (birth weight over 2,500 Gm.). Most were first-born, and were examined repeatedly. Labor lasted from 2 to 44 hours. The birth of 28 was spontaneous, with head as presenting part; 2 were delivered by forceps, and 3 by cesarean section. There were only 2 neonatal deaths, 11 days and 3 months after birth, respectively; both were examined postmortem.

The *general* and *focal* symptoms were the same as in the premature infants. The infant who died at the age of 3 months had manifested the most severe symptoms, bilaterally.

Spontaneous bilateral nystagmus of 4 to 8 weeks' duration was found on the first day of life in 25 infants. In many there was convergent or divergent strabismus. The results of hearing tests were like those of the premature infants.

Caloric stimulation elicited eyeball deviation in 16 infants, with the same course and direction as in Groups 1 and 2. The duration, however, was 30 to 60 seconds, and the latent period ranged from 20 to 30 seconds. Reexamination of several months later showed that the deviation had been replaced by typical nystagmus. In 12 infants, caloric stimulation soon after birth produced a typical nystagmus. In 3 infants, 2 of whom had the most severe clinical signs of birth injury, there was no response to caloric stimulation.

The rotatory test in all 3 positions gave the same results as in the other groups. Average duration of postrotatary nystagmus was 20 to 30 seconds.

The neurologic examination revealed normal skin and tendon reflexes in all infants.

Unilateral facial paralysis was found in 3 infants; in 2 the paralysis was undoubtedly of central nervous system origin, severe clinical signs of birth injury and sporatic convulsive seizures on the same side as the paralysis also being present. The third infant had been delivered by forceps, so that forceps injury could not be excluded. This infant, too, had various central nervous system disturbances: paresis of the abducens nerve, the head permanently turned to the right in tonic spasm, the right arm stretched out, and the left arm bent; with short interruptions, the tonic position was maintained for more than a week, and could be interrupted only for a few minutes by overcoming strong resistance. The manifestations strikingly resembled those described by Magnus and de Kleyn[57,58] in experiments on the neck reflexes.

TABLE 1. A FEW TYPICAL CASES OF BIRTH INJURY IN WHICH AUTOPSY CONFIRMED THE CLINICAL DIAGNOSES

CASE NO. 1: *Clinical Observations:*

Twin brother died at age of 15 days; birth wgt. 1,700 gms.; intense reddish color of skin the first day after birth; edema of extremities; spontaneous nystagmus. Three days after birth: child became very pale, icteric; asphyctic attacks followed; superficial respiration; no sucking reflex; lack of swallowing movements; very low body temperature; somnolence; child gave short, loud cries for no apparent reason; frequent yawning; rigid musculature; athetotic motions; progressive weight loss. At 10 days: mask-like expression, asphyctic episodes; persisting spontaneous nystagmus; caloric tests of vestibular apparatus negative; rotatoric excitation resulted in very poor response; no reaction to sounds; lumbar puncture gave intensely yellow-colored fluid containing shadows of red blood cells; muscle rigidity, singultus, yawning. Eleven days after birth: temperature 38.5°C (101.4°F.); respiration superficial; facial expression old and drawn.

Date of death: At the age of 11 days.

Autopsy findings:

Large number of birth-traumatic softening foci, as well as punctiform hemorrhages in both cerebral hemispheres.

CASE NO. 2: *Clinical observations:*

Birth wgt. 1,320 gms. First day after birth: hands and feet edematous; skin red; body temperature low. Second day after birth: vomiting causing feeding difficulties. Third day after birth: child did not hear; spontaneous nystagmus; vestibular irritability low; differences in vestibular reactions on both sides. Eight days after birth: frequent vomiting; apathy; severe icterus; general cyanosis; athetotic movements; sopor; muscle rigidity; singultus.

Date of death: At the age of 9 days.

Autopsy findings:

Multiple foci of pial hemorrhages; thick layer of these covering the cerebellum; large terminal vein hemorrhages had destroyed parts of caudate nuclei bilaterally; the white matter of both cerebral hemispheres was extensively disintegrated.

CASE NO. 3: *Clinical observations:*

Birth wgt. 1,300 gms.; 11½hrs. labor. On first day: low body temperature, asphyctic, apathic. On third day: subnormal temperature, asphyctic episodes; muscle rigidity, athetotic movements, yawning; feeding difficulties; intense spontaneous nystagmus; caloric reaction normal; rotatoric response only slightly below standard. Seven days after birth: icterus, vestibular reactions unchanged. Thirteen days after birth: progressive weakness, cyanosis, pallor of face, mask-like expression. Sixteen days after birth: bronchopneumonia.

Date of death: At the age of 16 days.

Autopsy findings:

Hemorrhages and hemosiderinic pigmentation of pia, especially intense on cerebellum; terminal vein hemorrhage on left; vein of Galen intensely dilated and filled with thrombotic masses; extensive softening had destroyed large areas of both cerebral hemispheres.

CASE NO. 4: *Clinical observations:*

First child of an 18-year-old woman; born after 21 hrs. hard labor; birth wgt. 2,940 gms.; cephalic presentation. First day of life: slight icterus; conjunctival hemorrhages in both eyes; scleroderma of extremities; twitching in facialis area; spontaneous nystagmus. Sixth day of life: feeding difficulties; frequent vomiting; progressive loss of weight; vestibular hypoirritability. Ninth day: general decline, continued loss of weight; intense pallor of face; general cyanosis; considerable fluctuation of body temperature. Tenth day: child emitted short, loud cries for no apparent reason.

Date of death: At the age of 11 days.

Autopsy findings:

Large hemorrhages of left cerebral hemisphere; extensive rarefaction (diffuse devastation process) of white matter in both cerebral hemispheres with numerous small cavities.

Group 4. The age of the 97 members of this group ranged from 6 days to 21 years. Weight at birth was unknown. All were examined repeatedly. The diagnosis on hospital admission was: (*1*) spasmophilia in 13; (*2*) mongolism and idiocy in 59; and (*3*) various diseases in 25. During the study, 4 of the children died.

(*1*) The 13 spasmophilic children were first examined at the ages of 3 months to 2 years; in 12 of them the clinical features were similar; 1 showed no signs of spasmophilia. Bilateral spontaneous nystagmus was present in 4 children. Repeated caloric tests with ice-cold and with warm (120° F.) water elicited no reaction in 12. Rotatory tests in all 3 positions showed slight positive results. Postrotatory nystagmus lasted 5 to 10 seconds only. Except in 2 children with a positive Babinski sign, the reflexes were normal.

It is noteworthy that, aside from the spasmophilia, spontaneous nystagmus was present in 4, that none of the children showed reactions to caloric stimulation, and that rotation elicited a weak, short nystagmus. This lack of reaction resembled that of younger children with birth injury, but had not been reported for this age group.

(*2*) The 59 idiots and mongoloids were divided into 2 groups: 5 under the age of 2 years, all mongoloids; and 54 persons 2 to 21 years old. Spontaneous Bilateral nystagmus was found in 3 of the 5 mongoloids; repeated caloric stimulation produced no reaction in any of the 5, and the response to the rotatory test was definitely slighter than normal in 4, and at times completely lacking. The reflexes were normal except for a positive Babinski sign in 2.

The noteworthy features in the history of the 54 others were: difficult labor, large scalp hematoma at birth, and poor physical and mental development. Little information could be obtained about hereditary or familial disorders. In many, convulsive seizures were said to have been present from birth, and spastic contractures, defec-

tive speech, facial tics, athetotic movements of the extremities, atrophy of an extremity, claw hand, hemiplegia, or paraplegia were present. The typical picture of Little's disease was found in some, while in others there were various endocrine disorders, such as myxedema or dystrophia adiposogenitalis.

Spontaneous nystagmus was found in 13 of the 54 patients. More than 50 per cent of the 54 did not respond to caloric tests; the reactions of the rest were normal.

The rotatory test could be performed only in the upright position. A 30 second nystagmus occurred in the great majority; the few whose nystagmus was of much shorter duration or absent, were without reaction to caloric stimulation.

The skin and tendon reflexes were extremely varied: normal, increased, or decreased patellar reflex; in many, a positive unilateral or bilateral Babinski sign, as well as a positive unilateral or bilateral Oppenheim sign and Gordon reflex, and foot and petellar clonus. In some, the neck and labryinthine reflexes were pathologic.

In a few patients, there was temporal pallor of the pupils, and partial or complete optic nerve atrophy.

(*3*) In the 25 patients admitted for a great variety of conditions, there were 2 with hydrocephalus, 2 with congenital syphilis, 1 with chorea, and 1 with encephalitis; 19 had various infectious diseases and nutritional disorders.

Only in the infants of this group which came to the hospital during the first few weeks of life did we find general symptoms which could be ascribed to birth injury; superficial respiration, muscular rigidity, pallor, athetotic movements, and convulsive seizures. Unilateral or bilateral spontaneous nystagmus was present in 8. In 16 the response to caloric stimulation was normal; in 5 there was no reaction, but 2 of them had recovered from meningitis and it is known that there is a lack of response in such cases. None of the 5 responded to the rotatory test; all the rest had a normal reaction. Unilateral facial paresis was present in 2.

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Part II of this paper, in a subsequent issue will contain a review of the literature, discussion and references

IDENTIFICATION OF NEWBORNS

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Increased interest has been displayed recently by many people regarding proper identification of newborn babies. And since identification of the newborn by sole prints at present is apparently considered sufficiently reliable by some health authorities to make their taking mandatory, it seems worthwhile to record the experience of the Woman's Hospital, Division of St. Luke's Hospital,* New York City, in a study made 12 years ago, but not previously reported.

Since 1946 many hospitals have used a triple method of identification.

First —neck beads on the baby and beaded bracelet on the mother.

Second—foot prints of baby and thumb prints of the mother in the delivery room.

Third —a picture of the baby in the nursery before discharge.

The most recent report was published in *Journal of Pediatrics*, January, 1957 by Norma Ford Walker, Ph.D.,[1] whose article is entitled "Inkless Methods of Finger, Palm and Sole Printing." At the conclusion of the article she asserts that it is difficult to get sharp prints with young infants.

According to F.B.I. Law Enforcement Bulletin of January, 1945,[2] footprinting for infants should provide the three following features: 1. individuality, 2. continuity, 3. immutability. With these requirements in mind the decision was made to sole and palm print 100 babies in the delivery room directly after birth. The same procedure was followed again 5 days later. Then the 2 sets of prints of each baby were identified and compared.

To eliminate technical error, one doctor was trained in the proper procedure of sole and palm printing by Mr. G. Tyler Mairs, a finger print expert. The author, after this preliminary training, took all the prints of the 100 babies selected, exercising extreme care during the process.

The method used was as follows: The baby's foot was wiped off with a clean piece of gauze and a thick printer's ink was applied to a

* *Acknowledgement is made to John Fitch Landon, M.D. at whose suggestion this study was undertaken*

glass plate. The infant's sole was pressed against this inked plate and then pressed onto special paper used for finger printing. The palm of the hand was treated in the same manner. The palms were more difficult to do because of the newborn's normal fist clenching. All the prints were then studied by Mr. Mairs, the expert on print reading.

Identification by palm and sole printing depends on the type whorl one has in his skin. Therefore, the report of the reading of the prints was interesting. No identifying whorls could be obtained from the prints made in the delivery room. However, the whorls were identifiable on the prints taken at 5 days of age. The results, according to our studies, showed there would be no positive identification of the individual newborn's sole and palm prints made in the delivery room.

After much thought and discussion it was felt that the amount of vernix on the skin was probably a contributing factor in our not getting the identifying whorls in the new born and that perhaps a further study would be helpful if some solution could be used to cleanse the sole and palm in the delivery room before making the prints.

The claim has been made that identification can be determined by the creases on the sole prints. In view of these statements, the author reviewed all the prints of the 100 newborns previously taken at Woman's Hospital. The findings were that the creases on the sole and palm prints were not the same in the delivery room as those taken 5 days later. Hence, the reading of creases seems inadequate also.

As a result of the experiment at Woman's Hospital in 1946, Mr. Mairs and the author felt that there were two ways to obtain a positive identification of the newborn. The first one was to find some solution that could be used to cleanse the skin before taking the sole and palm prints in order that better identification might be made. The difficulty encountered with this suggestion was that solutions used should not be irritating to the newborn's skin. Peroxide, mineral oil, soap and water, and a detergent solution were tried. The detergent solution was found to be most efficacious for removing the vernix.

At the present time, the second way that positive identification could be made is to photograph the newborn's ears. It has been

noted that ear configurations do not change. This is a study now under consideration in the delivery rooms and newborn nurseries at Morristown Memorial Hospital, Morristown, New Jersey. We shall report on the results in the near future.

SUMMARY

The difficulties of obtaining palm and sole prints which would serve as positive means of identification of the newborns has been discussed. It is further suggested that new and surer methods are desired. A suggestion that is being studied at the present time is one concerning the identification of newborns by photographs of ears.

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To the Editor
Archives of Pediatrics
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September 24, 1959

With the increasing importance of perinatal infections due to Listeria monocytogenes in the human subject, the recent paper by Dr. Milton O. Kepler[1] and his letter in the May, 1959, issue of ARCHIVES OF PEDIATRICS *deserves more than passing consideration. For the past several years my major research efforts have been directed toward infections due to Listeria monocytogenes in both man and animals and I have watched with considerable interest the development of the increased awareness of the importance of these infections. It is the opinion of many of us who have worked closely with this bacterium that listeric infections are not actually rare, but rather that they are unrecognized. It appears to be more than mere coincidence that in many instances after the first case is reported from a certain district or laboratory, others are reported within a relatively short time. In addition to those cited in Dr. Kepler's letter, one could mention the experience of Dr. Hood, Charity Hospital, New Orleans who has observed some 25 cases;[2] Dr. Welshimer, Medical College of Virginia, Richmond, eight cases;[3] Dr. Patocka and co-workers, Charles University, Prague, Czechoslovakia, more than 40 cases;[4] Dr. Potel, formerly of the Martin Luther University, Halle, East Germany, 85 cases;[5] Dr. Trüb, District Medical Director, Düsseldorf, West Germany, 60 cases,[6] and my own experience while at Michigan State University where we confirmed the identity of 12 cultures from human origin, only two of which have been published.[7] This list is by no means exhaustive, and many other examples could be given. Of significance for the pediatrician and obstetrician is that of the above cases more than 75% involved neonates.*

Several factors contribute to the lack of awareness of infections due to L. monocytogenes. Among these are the misconception that listeric infections are rare; the bacterium's resemblance to some of the common

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This department is for readers' views and observations. The editors will welcome reports or letters of pediatric and related-subject significance.

gram positive diphtheroids results in its being discarded as a contaminant; and the difficulty of isolating it from certain tissues with the consequence that the culture is reported as negative. Although it is true that L. monocytogenes grows well on most of the commonly employed bacterial media, it is not true that it can always be cultivated easily from infected tissues or body fluids. More than 10 years ago Gray et al[8] *described a method for enhancing the probability of isolating L. monocytogenes based on maceration and refrigeration of the tissue to be cultured. During the intervening years the reliability of this method has been confirmed by numerous investigators in several different countries working with both naturally and artificially infected subjects. This technique has been used with good success on tissues from human neonates in the Prague area,*[9] *and recently Kranendonk*[10] *showed that vaginal swabs taken on the 10th and 21st postpartum day from a woman who had given birth to an infected infant revealed the presence of L. monocytogenes only after they had been refrigerated for several days. Refinements of this technique has been published recently by Gray*[11] *and Bearns and Girard*[12]*. Although it must be admitted that this method has several serious disadvantages for the diagnostic laboratory, there is a feeling that if it were employed more widely, it would be found that listeric infections are more prevalent than is generally suspected. Listeric infection cannot be ruled out merely on the basis of a negative finding on primary culture attempts.*

The point which Dr. Kepler makes that listeric infection may be more prevalent among "lower economic brackets" deserves considerable emphasis. Although not all cases of listeriosis occur among this group, the distribution, as judged by both published and unpublished cases tends to bear this out. The first reports of real consequence of perinatal listeric infection in man originated in East Germany.[13] *The syndrome was being recognized with increasing frequency even before Dr. Seeliger of the Hygine Institute, University of Bonn, first suggested that this might be a listeric infection rather than an entirely new entity.*[14] *Even after the medical profession in both sectors of Germany became aware of the disease, the incidence remained strikingly higher in the eastern countries. Of the many talks on listeriosis which I gave in Europe while I was at the Justus Liebig University, Giessen, Germany on a Fulbright Research Grant, I often mentioned, if I felt that the audience was appropriate, that in contrast to man, Listeria monocytogenes seemed to enjoy more freedom behind the Iron Curtain than on this side of it. Further support for this contention is found in the 25 cases of listeriosis observed by Dr. Hood.*[2] *This is without doubt the largest number of cases observed in*

any one area in the U. S. Most of these were in neonates and a majority of the patients were either negro or "poor whites." Also in my own experience some of the patients were either transients, skid row alcoholics, prostitutes, or charity cases. The circumstantial evidence in favor of poor living conditions as a predisposing factor is very convincing. In contrast, among animals, usually the very best in the flock or herd are the ones affected.

Since the epidemiology of all forms of listeriosis is still obscure, one can only speculate on the possible relationship between economic level and susceptibility. It is possible that L. monocytogenes may commonly be present in soil, domestic animals, or some animal products.[15,16] *It is established that L. monocytogenes may be present in apparently normal individuals and that it may be responsible for many low grade flue-like or infectious monocleous-like infections,*[15,16,17] *which when they occur in the nongravid subject are of no serious consequence, but in the gravid subject invariably terminate in fatal infection of the fetus. It has been shown experimentally that subjection of laboratory animals to various forms of stress, including starvation, greatly enhances their susceptibility to listeric infections.*[17,18] *If low economic level produces sufficient stress to enhance host susceptibility to an often present pathogen, this may be a possible explanation for the relatively higher incidence among this group. The misconception that listeriosis is always an acute, highly fatal disease has done much to impede the unraveling of the epidemiology and pathogenesis of the various forms of this still poorly understood infection.*

It is known that recovery from listeric meningitis in neonates may result in permanent damage to the brain.[15,16] *Recently it has been suggested that if inapparent infections occur in the newborn, it may lead to either meningitis in the neonatal period or retarded mental development during childhood.*[15,19,20] *This latter is based on studies by Lang*[20] *which showed that among a group of mentally retarded children in which the etiology was not established, 43 out of 87 showed significant titers against L. monocytogenes while only 5 out of 50 children in which the etiology was established showed similar titers. Also Rost et al*[21] *have suggested that inapparent listeric infections may be responsible for some cases of habitual abortion in women. They found that some patients showed a marked rise in titer against L. monocytogenes at the time pregnancy was interrupted. When these patients were treated with a combination of tetracycline, penicillin and sulfonamides during the next pregnancy, they gave birth to apparently normal full-term infants. This*

method succeeded with 6 to 8 patients, some of which had had from 3 to 7 consecutive abortions. Other cases have been reported subsequently in Germany and Czechoslovakia.[15,16]

At present these observations on possible mental deficiency and habitual abortion have been based entirely on serological evidence and have not been confirmed by isolation of L. monocytogenes. However, the studies of Gray et al[15,22,23] *on artificially induced perinatal listeric infections in rabbits support these postulations. They found that some kindlings born to does exposed orally before or immediately after, or intravaginally during parturition experienced inapparent infections which could be detected only by isolation of the bacterium from the urine or at necropsy by isolation from the viscera and/or the brain of those sacrificed. Some apparently normal, lightly infected kindlings developed meningitis 5 to 7 days postpartum. Physically, the apparently normal kindlings permitted to survive and some which recovered following symptoms of a central nervous system disorder, developed normally, but in the absence of suitable tests there was no measure of mental development. Although most of the does which had given birth to infected litters delivered normal kindlings following the next gestation, a few aborted during the third week and L. monocytogenes was isolated from the aborted material. One doe suffered three consecutive abortions following one exposure. She remained sterile after the last abortion. Some apparently normal does which had given birth to infected litters had small abscesses containing L. monocytogens in the uterus when sacrificed one month postpartum. These abscesses could constitute a source of infection for the next litter. These findings simulated in almost every detail listeric infection in human neonates and support the postulation that this bacterium may cause mental retardation in young children and that it may be responsible for some forms of habitual abortion. Further investigation is required, but these findings suggest that listeric infections may play a more prominent part in disorders of man, particularly in the perinatal period, than has been suspected previously.*

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Sincerely,

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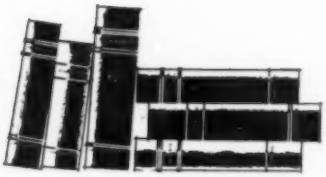

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Edited by

MICHAEL A. BRESCIA, M.D.

KLINISCHE METHODEN DER BLUTGERINNUNGSANALYSE, by Von Prof. Dr. J. Jurgens, Doz. Dr. F. K. Beller and von Prof. Dr. M. Gansslen. Cloth. Pp. 408. Price $13.35. Georg Thieme Verlag, Stuttgart, Germany 1958. In the U.S.A. Intercontinental Medical Book Corp., New York.

This volume has been dedicated by the authors to the memory of Paul Morawitz, founder of modern physiology of coagulation, whose classical theory still stands in spite of additions and modifications. The emphasis is on laboratory technique of which approximately two thirds of the book is devoted. In contrast to such highly individualistic presentations of the subject as those by Quick and Biggs and McFarlane which reflects the particular workers determined independance, this volume is a successful attempt at international synthesis. Not only are the authors thoroughly familiar with the work of American and British coagulationists but as one digests the book the unbiased reader becomes aware and appreciative of the numerous contributions made in this field by Continental European workers. This refers not only to the well-known work of Koller's group but also to that of a great number of others less well known in this country. The book is astonishingly up-to-date. Ample space has been alloted to the fibrinolytic system, the coagulation inhibitors and the position and determination of more recently detected factors such as the Hagemann and Stuart Prower factors which are discussed in a very lucid manner. There is also a short chapter on the calculation of clotting factors and one on thrombelastography.

The authors have succeeded in discussing a difficult subject without oversimplification on the one hand nor compounding the controversies on the other. Furthermore, the fact that no new synonyms are introduced for already familiar factors is a rarity and a merit in itself.

Thus, this volume has many fine features. The subject matter is well arranged and well written without excess wordage. Bibliographies are excellent. Anyone interested in coagulation work and knowing German well will find the book worthwhile reading.

ROBERT FELDMAN, M.D.

ANESTHESIA FOR INFANTS AND CHILDREN. By Robert M. Smith, M.D. Price $12. The C. V. Mosby Co., St. Louis 3, Mo.

In the words of the author "the main purpose of the book has been to assemble as much of the practical information available concerning the basic response of children to anesthesia." This is a book that should be of interest not only to anesthesiologists, pediatricians, but also to pediatric surgeons. He discusses problems that are related to all three specialties.

There is a wealth of knowledge and facts that will be of great use to the anesthesiologist who is called upon to anesthetize infants and children. He mentions the pitfalls and errors to avoid and gives a practical clinical approach to the various problems from his vast experience.

The chapter on Respiratory Physiology in Infants and Children by Dr. Charles D. Cook is an excellent summary of the present concepts on respiration and respiratory physiology.

We all know that the problem of pre-operative medication is a controversial subject but the author in his chapter on Premedication shows that the advantages are greater than the disadvantages provided care is used to avoid overdosage and depression.

Dr. Smith also makes the point that although much expensive and elaborate apparatus has been debeloped for observation of patient, uncomplicated devices are preferable for most circumstances. He also discusses the various problems with their management that face the anesthesiologist such as anesthesia for the newborn, use of hypothermia, cardiac surgery, plastic surgery, etc.

The chapters on Emergency Surgery and Fluid and Blood replacement are very lucid and should be recommended reading for all surgeons so that they may be aware of the problems which present themselves to the anesthesiologist.

There is no doubt that this book will serve as a ready reference for residents in Anesthesiology, anesthesiologist, and pediatric surgeons. There have been several other texts over the years on pediatric anesthesia but this book at the present time is the ultimate.

LOUIS S. BLANCATO, M.D.

BIOLOGICAL PSYCHIATRY. Edited by Jules H. Masserman, M.D., with 77 contributors. Pages 328, cloth, Grune and Stratton, Inc., 1959. Price $9.75.

This book represents the transactions of the Society of Biological Psychiatry at the San Francisco, May 1958 scientific sessions. The proceedings comprise physicians in the fields of neurology, psychiatry, psychology, neurophysiology, and neurochemistry. These sciences are correlated into the biological behavior of human beings as well as animals. The animal experimentations are numerous, rewarding, enlightening and encouraging. The experimental work on humans ranges from the anxiety states and personality patterns to the psychoses and schizophrenic phases. The drugs used in therapy range from the rauwolfia alkaloids, the amphetamines, ritalin, disipal, suavitil, etc., to a comparative study of the Phenothiazine tranquillizers. These include trilafon, Dartal, compazine, vesprin, thorazine, sparine and pacatal. The evaluation of drugs and their effects are taken into detailed consideration and study. Finally, there are over seventy-seven (77) contributors from all over the country reporting their results of research as related to psychiatric diseases.

The text is divided into five parts: Part I: Neuroanatomy and Neurophysiology, Part II: Clinical Research, Part III: Psychopharmacologic Research, Part IV: Drugs in Psychiatric Therapy, Part V: Evaluations and Discussions.

The clinical research section entails considerable physiology and biochemistry such as metabolic studies in the various psychoses, the catechol amines, proteolytic enzyme activity in cerebro-spinal fluid and the action of epinephrine breakdown products on cerebral functions, etc.

Biological Psychiatry is a comprehensive, valuable book for all psychiatrists, internists and general practitioners. The multifarious drugs on the market are now assembled and the effects discussed so that indications and contra-indications become well clarified. Psychotherapy and psychopharmacology are well combined, thus leading to the possibility of a comprehensive approach to mental diseases and disorders with a co-ordinated therapeutic orientation.

JOSEPH M. COVELLI, M.D.

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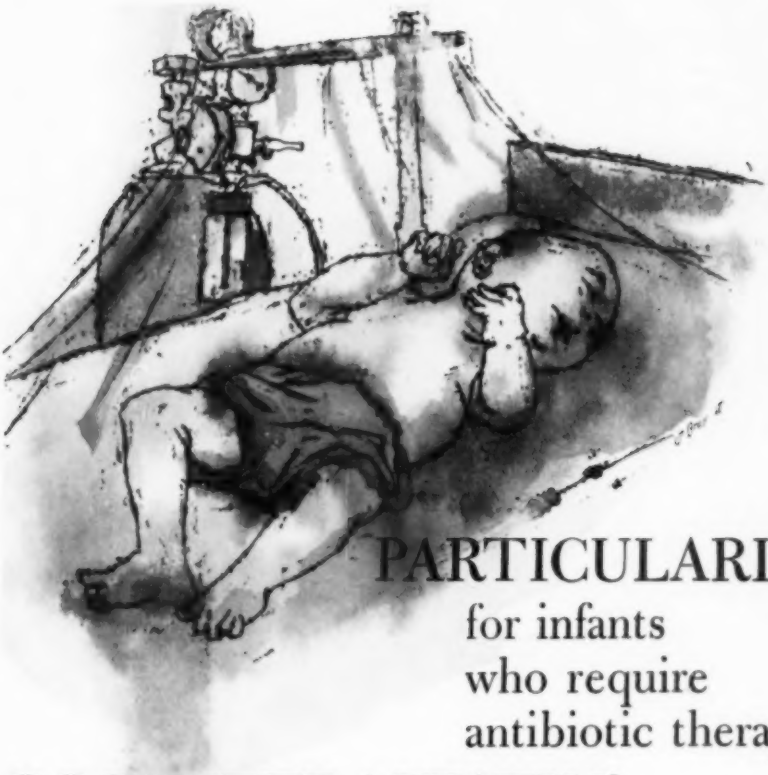

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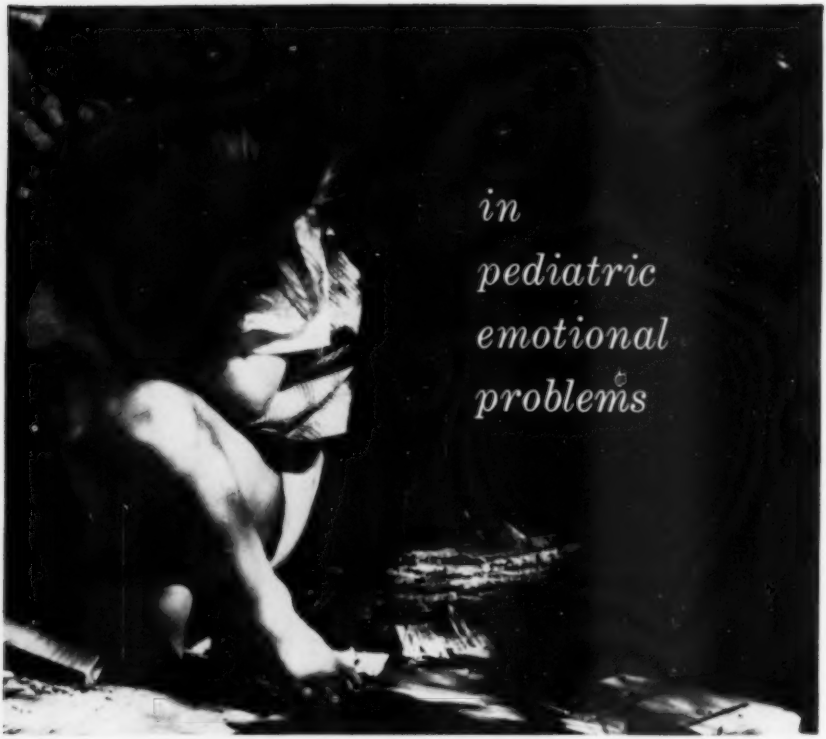

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